



AHL Newsletter

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June, 2013

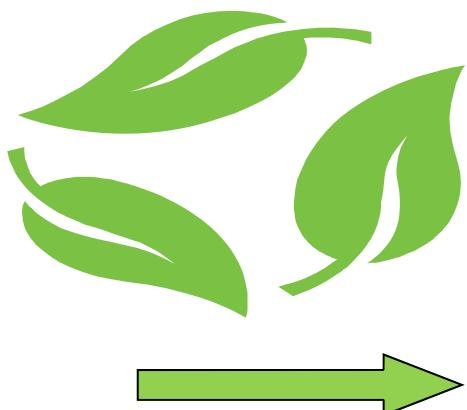
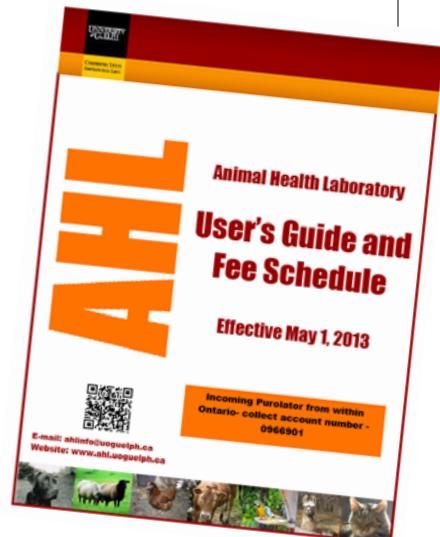
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In this issue:

2013 User's Guide and Fee Schedule	9
Going green!!	9
Mastitis workshop, 2013	10
Influenza testing options	11
VTM available	13
Ruminants	
Bovine rotavirus corona-virus diarrhea	12
Swine	
<i>M. hyopneumoniae</i> tests	13
<i>Brachyspira</i> update	13
VTM available	13
Avian/fur/exotic	
Torsion of hepatic liver lobe in a rabbit	14
Horses	
<i>Klebsiella</i> septicemia	15
Equine abortion summary	15
VTM available	13
Companion animals	
Necrotizing fasciitis	
<i>Streptococcus canis</i>	16

May 1, 2013, AHL User's Guide and Fee Schedule

- Mailed mid-April - we would be happy to send clients additional printed copies - the User's Guide and Fee Schedule are both available on-line.
- View and search available tests at <http://www.ahl.uoguelph.ca>—click on Fee Schedule.
- To log-in for fees, for Vet-clients only - contact us at 519 824-4120 ext. 54320 or at ahlinfo@uoguelph.ca for the **Client Access** username and password.
- **Results are available direct from our LIMS (Lab Information Management System)** – please contact us for further details ahlinfo@uoguelph.ca
- **Updated pricing** effective May 1, 2013 to April 30, 2014. Some fees have been increased to keep up with inflation, but some have been decreased as a result of technological and process improvements, e.g., gene sequencing has been reduced from \$200 to \$100.
- Bacteriology testing update: Please note that “Bacterial culture, **fecal culture**, companion/other - Includes detection of *Salmonella* spp., *Campylobacter* spp., *Yersinia* spp., and *Clostridium perfringens*, *C. perfringens* enterotoxin by ELISA, and susceptibility for aerobic bacteria only.”
- **Out-of-province pricing** for most food animal tests is set at 150%, but this rate has been adjusted for some tests, and out-of-province fees are now listed in the on-line Fee Schedule.
- The term “necropsy:” has been replaced throughout with “postmortem”, as part of the current trend in veterinary pathology. *AHL*



We're going green!!

We plan to distribute the June, 2013, issue of the AHL Newsletter as our last mailed hard-copy - we will then migrate to electronic publication. The AHL Newsletter has been available on-line for several years, and is easily accessible at <http://guelphlabservices.com/AHL/Newsletters.aspx>

Notification and Table of Contents of each newsletter will be sent to all addresses currently on our email distribution list - click on any topic of interest. **If your practice is on the list, but you would like to receive a personal notification, please send your request to:**

holiver@uoguelph.ca

We welcome your feedback.

AHL hosted the second “Diagnostic Bacteriology for In-Clinic Laboratories Workshop” on April 23rd 2013

Durda Slavic, Ann Godkin, Jim Fairles

Dairy veterinarians are always looking for improved ways to establish protocols for treatment of clinical mastitis. The emphasis is always on prudent use of antimicrobials and if they are not needed, they should not be used. Many organisms that cause clinical mastitis in dairy cattle are not amenable to treatment, and it behooves the veterinarian to find ways to make optimal treatment decisions. These decisions need to be very timely and an answer as to whether to treat or not should be made within 24 hours. From a diagnostic laboratory standpoint, these timelines are difficult to meet, especially if distances are involved and an overnight courier is needed. An in-clinic (or on-farm) milk bacteriology laboratory is one of the ways to handle this challenge.

Seventeen veterinarians and technicians from several clinics in Ontario met at AHL-Guelph on April 23 for our second training session on diagnostic clinical bacteriology of milk. **Jim Fairles** gave an overview of the milk collection process and the need for proper sterile technique in sampling. In any type of bacteriology, this is one of the most important steps! **A good sample is a must for good results.**

Ann Godkin, from OMAF and MRA, provided an overview of in-clinic diagnostic labs and of the supplies and procedures that would be needed. There are a multitude of considerations to make “beyond the plates”. One of the major areas to consider is quality assurance and how the in-clinic lab can, on an ongoing basis, verify that the results they are giving to clients are assured to be the correct ones.

Durda Slavic, AHL veterinary bacteriologist, then led the group through some diagnostic bacteriology discussions and hands-on work on basic milk bacteriology. Demonstrations showed the 3 main types of plating techniques that are used in most in-clinic laboratories - **traditional plating, Minnesota Triplates, and 3M Petrifilm**. Each clinic had a homework assignment in which they plated milk at their lab and sent in a split sample to the AHL. These were compared and discussed as well.

Feedback was greatly appreciated after the meeting as we move forward on providing more outreach opportunities for our veterinary clients. AHL would like to thank the Animal Health Strategic Investment for partial funding of the exercises, OMAF and MRA for Dr. Godkin's input and the clicker technology, as well as 3M and the University of Minnesota for handout material. *AHL*



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Its mission is to inform AHL clients and partners about AHL current activities, and laboratory-based animal disease events and disease trends. All material is copyright 2013. Ideas and opinions expressed herein do not necessarily reflect the opinions of the University or the Editor.

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Our continued thanks to all of the non-author AHL clerical, technical, and professional staff who contribute to the generation of results reported in the AHL Newsletter.

Summary of *Influenza A virus* testing at the AHL, May 1, 2013

STEP 1				VIRUS DETECTION TESTING				ANTIBODY DETECTION TESTING			
Sample	Swabs in VTM (swine-nasal, birds-cloacal/tracheal), oral fluids, tissues	Serum		Test	Real-time PCR - matrix gene	ELISA, Multi-Screen	Agar gel immunodiffusion	Test			
Cost	\$28.00 (can be pooled to decrease the cost)	\$5.00 (do not pool)	\$3.40	Use/advantage	Primary screening test targeting a conserved influenza virus gene. Detects all common <i>Influenza A virus</i> subtypes in multiple animal species, very sensitive, fast.	Primary screening test, detects antibody from all common <i>Influenza A virus</i> subtypes in multiple animal species.	Screening test used for avian samples.	Dissadvantage	Cannot determine the subtype of the virus.	Cannot determine the subtype of the virus to which animals were exposed. Cannot be used on paired samples to determine a 4-fold titer change/seroconversion.	
STEP 2				VIRUS SUBTYPING				ANTIBODY SUBTYPING			
Sample	PCR positive sample	Hemagglutinin gene sequencing	ELISA, H1N1 (swine only)	Test	PCR typing for specific subtypes	ELISA, H1N1 (swine only)	ELISA, H3N2 (swine only)	Test	Serum	Hemagglutination inhibition test (H1N1 & H3N2)	Hemagglutination inhibition test (H1N1 & H3N2)
Cost	\$28.00 per H subtype	\$100.00	\$9.00	Use/advantage	Swine: H1N1 & H3N2 Turkeys: H5& H7, also H1N1 & H3N2 All other avian species: H5 & H7 Simpler, faster than sequencing.	Covers all common <i>Influenza A virus</i> subtypes. Allows strain identification and comparison of various viruses.	Use to determine if animals were exposed to H1N1 subtype.	Detects antibodies only to H1N1 subtype. Cannot be used on paired samples to determine a 4-fold titer change/seroconversion.		\$9.00 per subtype	Use on paired samples to determine a 4-fold titer change/seroconversion.
Dissadvantage	Weak positives need virus isolation to produce enough virus for typing. Detects only H1N1, H3N2, H5 & H7.	Weak positives need virus isolation to produce enough virus for typing.	Detects antibodies only to H1N1 subtype. Cannot be used on paired samples to determine a 4-fold titer change/seroconversion.								
OTHER TESTS											
Sample	Nasal swabs in VTM, tissues	Formalin-fixed tissues		Test	Virus isolation in embryonated eggs, virus isolation in cell culture	Immunohistochemistry		Cost	\$31.50 (flock animal), \$50.00 (companion/other)		
Use/advantage	It may be required to produce virus for typing. Detects common <i>Influenza A virus</i> subtypes.	Used on fixed tissues when fresh tissues are not available, or as a part of postmortem procedures. Detects all common <i>Influenza A virus</i> subtypes.	Dissadvantage	Time consuming, not as sensitive as PCR. Some viruses cannot be easily propagated in eggs or cell culture. Cannot determine the virus subtype.							

Prepared by K. Harron, J. Fairles and D. Ojikic. For further information or to request tests please email ahl.virology@uoqueph.ca

AHL Lab Reports

RUMINANTS

Diagnosis of bovine rotavirus and coronavirus infection in calf diarrhea

Andrew Brooks, Josepha DeLay, Davor Ojkic

Diarrhea is a common clinical problem in young dairy and beef calves. Rotavirus, *Bovine coronavirus*, *E. coli* and *Cryptosporidium* are frequently detected in calf diarrhea cases at the AHL (Table 1). Rotavirus and coronavirus infections cause damage to the intestinal epithelial lining and atrophy of the intestinal villi, which results in diarrhea, dehydration and electrolyte losses. This article summarizes the diagnostic test options for rotavirus and coronavirus infections in cases of calf diarrhea (Table 2).

Tests for rotavirus and *Bovine coronavirus* involve detecting viral antigen or nucleic acids in the feces or the intestine. From live animals, early in the disease process, submit fresh, chilled or frozen diarrheic feces. From dead animals, submit chilled or frozen, as well as formalin-fixed segments of spiral colon, ileum with Peyer's patches, and jejunum. Sacrificing a live, acutely ill, untreated calf for postmortem at the AHL provides the best opportunity to reach a diagnosis. A comprehensive guide to sample submission for diarrhea cases is also available in the AHL User's Guide.

The ***Bovine coronavirus* antigen ELISA (BCE)** and **rotavirus group A latex agglutination test (RLA)** detect viral antigen in the feces or intestinal contents. The BCE and RLA tests are relatively inexpensive and have historically been used as primary screening tests for individual animals. The BCE and RLA tests are not recommended for pooling of samples from multiple animals.

In more recent years, real-time **polymerase chain**

reaction (PCR) tests that detect rotavirus and coronavirus nucleic acids have been developed and adopted by the AHL. These PCR tests have short turnaround times and can be performed on fresh feces, intestinal contents, or segments of jejunum, ileum and colon. These tests tend to be more sensitive, so pooling samples from multiple animals (up to 5) can be done to decrease the cost of testing. The rotavirus PCR test detects both type A and B rotaviruses, in contrast to the RLA (rotavirus type A) test.

Immunohistochemistry (IHC) is available at the AHL for both *Bovine coronavirus* and rotavirus. IHC is performed on the same block of formalin-fixed, paraffin-embedded tissue used for histologic examination, and this test method allows direct correlation between histologic lesions and presence of viral antigen (Figure 1). IHC for infectious agents is generally less sensitive than PCR and, as a result, is not typically recommended as a first-line test for coronavirus and rotavirus. However, if only formalin-fixed tissue is available for a case and lesions are compatible with viral enteritis, IHC can be a very useful test to confirm the etiology. IHC can also be useful in cases with histologic lesions of viral enteritis but for which other microbiologic tests have not confirmed viral infection. Use of IHC for confirming the presence of these enteric pathogens is most successful when tissues are collected and placed in formalin immediately after euthanasia. This sampling method ensures preservation of potentially virus-infected epithelium covering intestinal villi. *AHL*

Table 1. Common infectious causes of calf diarrhea.

Pathogen	Age range (J,K&P*)	Age range (Radostits **)
<i>E. coli</i> (ETEC)	≤ 7 days	≤ 3 days
<i>Bovine coronavirus</i>	≤ 7-14 days	5-21 days
Rotavirus	≤ 7-14 days	5-15 days
<i>Cryptosporidium</i>	4-20 days	5-35 days
BVDV	≥ 7 days	≥ 14 days
<i>Salmonella</i> spp.	≥ 4-5 days	5-42 days
BHV-1 (IBRV), alimentary form	≤ 14 days	< 10 days
Other viruses (Breda virus, bovine parvovirus, etc.)	various	14-30 days

*Maxie, MG. Jubb, Kennedy & Palmer's Pathology of Domestic Animals, 5th ed., 2007.

**Radostits O, et al. Veterinary Medicine, 10th ed., 2007.

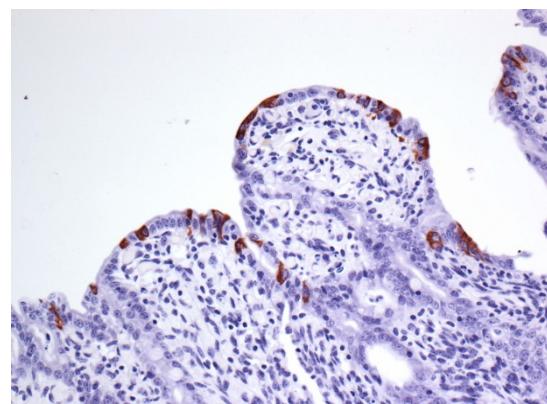


Figure 1. Bovine coronavirus IHC, calf small intestine. Red-brown cytoplasmic staining represents *Bovine coronavirus* antigen in atrophic villi.

Diagnosis of calf diarrhea, continued

Table 2. Summary of diagnostic tests for bovine rotavirus and coronavirus at the AHL

Test	Sensitivity	Cost	Sample type	Pooling of samples	Turnaround time
BCE	+	+	live: feces; dead: intestinal contents, colon, ileum, jejunum.	Not recommended	7-14 days
RLA					
IHC	++	++	formalin fixed colon, ileum, jejunum	Not recommended	1-10 days
Real-time RT -PCR	+++	++	live: feces; dead: intestinal contents, colon, ileum, jejunum.	Yes – up to 5 animals	3-7 days

SWINE

Mycoplasma hyopneumoniae testing update Hugh Cai

Effective March 6, 2013, we have been automatically repeat-testing sporadic *Mycoplasma hyopneumoniae* IDEXX ELISA suspicious/positive samples with Oxoid Myo ELISA kits on Wednesdays and Fridays. As well, to increase the consistency of the ELISA results, we have ordered a large quantity of the kits with the same lot number, and we test them against negative serum before use. *AHL*

Brachyspira update, January to April 2013 Durda Slavic

Since January 2013, the AHL has investigated 25 cases submitted for *Brachyspira hyodysenteriae* and *Brachyspira pilosicoli* PCR testing. In total, 68 samples were included in the investigation including 19 tissue samples and 49 fecal samples. All samples were tested for the presence of *Brachyspira* spp. by culture using 2 different selective media and by real-time genus-specific *Brachyspira* PCR.

On the sample level, 11/68 (16.2%) samples were positive for *Brachyspira* spp. by culture, and 24/68 (36.8%) were positive by real-time PCR. All PCR-positive samples were further tested for the presence of *B. hyodysenteriae*, *B. pilosicoli*, *Brachyspira intermedia*, *Brachyspira innocens*, *Brachyspira murdochii*, *Brachyspira hampsonii* clade I and *Brachyspira hampsonii* clade II by real-time PCRs. No *B. innocens*, *B. hampsonii* clade I or *B. hampsonii* clade II were detected in any of the samples investigated. *B. hyodysenteriae* was detected in 14/24 samples, *B. pilosicoli* in 7/24 samples, *B. intermedia* in 5/24 samples, and *B. murdochii* in 9/24 samples by real-time PCR. *B. pilosicoli* was always detected together with *B. hyodysenteriae*, whereas *B. intermedia* was

detected either in combination with *B. hyodysenteriae* and *B. pilosicoli* or on its own.

At the case level, **only 4 cases of *B. hyodysenteriae* were confirmed**. Two of these cases originated from 2 farms in close proximity operated by family members, whereas the 2 other cases were submissions from a farm that is geographically distant from the family-operated farms. In addition, **2 cases were positive for *B. intermedia* and 3 cases were positive for *B. murdochii* over a 4-month period**. At present, the significance of *B. intermedia* and *B. murdochii* detection remains to be established.

We will continue to investigate any swine dysentery suspect cases to monitor the potential introduction of *B. hampsonii* clade I and clade II isolates into the Ontario swine population. **If you suspect swine dysentery, please submit feces or tissue samples to the AHL for *Brachyspira* culture and PCR.** Call the laboratory in advance to alert us that samples are coming, since a selective medium for culture is made on an as-needed basis. *AHL*

Virus transport medium available from AHL for viral disease testing

When diagnosing viral diseases (e.g., for Equine practitioners - *Equine herpesvirus 1 / EHM* or *Influenza A virus*) it is very important that AHL receives the proper sample in the proper medium in order to obtain the best result. For AHL clients, VTM can be purchased from AHL (small quantities) at \$2.00 per vial. It is also available directly from: **VWR**, Starplex Multitrans Collection and Transport System (for transportation of viruses, chlamydia and mycoplasma) # S160-100 (case of 100) (Cat CA73270-008). Each tube contains transport medium and one swab. <https://ca.vwr.com/> **Fisher:** B220221 \$103.46 UNIV VIRL TRANS SWAB STD 50/PK.

AVIAN/FUR/EXOTIC SPECIES

Acute torsion of the hepatic quadrate lobe in a mature pet rabbit

Marina Brash, Gillian Park

Acute torsion of a liver lobe can clinically mimic gastrointestinal stasis, a more commonly recognized condition in rabbits, and should be considered in the list of differentials when rabbits are presented with lethargy, anorexia and abdominal pain of short duration.

A 3-year-old neutered male Florida White rabbit weighing 1.54 kg was presented for clinical evaluation. The owner described the rabbit as being lethargic and uncomfortable, with cold ears. The rabbit had not eaten or produced any fecal pellets for over 24 hours, but was still drinking. The owner described the rabbit appearing to be repulsed by food. There was no known ingestion of foreign material such as carpet or fabric. The rabbit had experienced previous episodes of GI stasis.

At the time of presentation, the rabbit was depressed and lethargic with slightly pale mucous membranes, heart rate of ~250 bpm, normal respiration, rectal temperature of 37.8°C, however the ears felt slightly cold. The stomach was mildly distended with fluid ingesta and a moderate amount of gas was present in the cecum. Supportive treatment was instituted that included subcutaneous fluids, pain and anti-gas medications, and force feeding of a nutritional supplement (Critical Care, Oxbow Animal Health, Murdock, NE, USA), papaya enzyme, vitamins B and C.

The rabbit did not improve with supportive care, so diagnostic testing was conducted. The ALT was markedly increased (932; reference interval (RI): 15-74 U/L); amylase was moderately increased (2,237; RI 100-1,100 U/L), urea and creatinine were markedly elevated (urea 46.3; RI 6.1-8.6 mmol/l, creatinine 824; RI 70-159 mmol/L) and glucose was slightly reduced (4.9; RI 6-8.9 mmol/L). Because of the lack of clinical improvement and the biochemistry results indicating severe hepatic and renal damage, the owner elected euthanasia and gave permission for a postmortem exam to be conducted.

At **postmortem**, the quadrate liver lobe was enlarged and hemorrhagic and there was a small amount of bloody fluid in the abdomen. No foreign material was identified in the gastrointestinal tract. All other organs, including the other liver lobes, were within normal limits. The tentative diagnosis was torsion of the quadrate lobe of the liver.

Histologically, the affected liver lobe was markedly congested and hemorrhagic with generalized acute parenchymal necrosis sparing the bile duct epithelium, and with fibrinous thrombosis of multiple vessels. Necrotic neutrophils were clustered multifocally within sinusoids and the capsular surface was coated with a layer of fibrin, red blood cells and viable neutrophils. These changes are consistent with acute hepatic infarction secondary to hepatic lobe torsion. There was marked acute renal tubular necrosis with hematuria,

proteinuria and hemoglobinuria; there was also very early tubular epithelial regeneration and mild multifocal nonsuppurative interstitial nephritis.

The rabbit liver has 5 lobes - the right lobe, the quadrate lobe located behind the gallbladder, a small caudate lobe near the right kidney, and the left lateral and left medial lobes. In Figure 1, the quadrate lobe is enlarged and rounded with large dark areas of hemorrhage and dark red exudate on the capsular surface. Contrast this affected quadrate lobe with the image of a normal quadrate lobe (Figure 2).

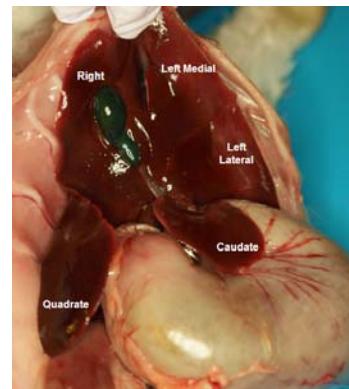
There are published reports describing torsions of the right, quadrate and caudate liver lobes and in most cases the diagnosis is made at postmortem. However, one report describes torsion of the caudate lobe as an incidental finding in 3 research rabbits dying with acute pasteurellosis. Because of the difficulty in clinically diagnosing liver lobe torsion, clinicians resort to exploratory laparotomies and in 2 recent reports, torsion of the caudate lobe was identified and the lobe resected. One rabbit survived the surgery, but the other rabbit died during recovery from anesthesia.

Torsion of the quadrate lobe in a pet rabbit is an infrequently reported cause of lethargy, anorexia and abdominal pain, and should be considered in cases with elevated liver enzymes. Clinical diagnosis is challenging and surgical resection must be performed early to prevent death from the toxic products of hepatic necrosis. Clinical chemistry evaluation is indicated at presentation to effectively rule out this condition. *AHL*

Figure 1. Swollen hemorrhagic quadrate liver lobe with exudate on the capsular surface (green arrow).



Figure 2. The normal rabbit liver consists of 5 lobes - the right lobe with the gallbladder, the quadrate lobe located behind the gallbladder, a small caudate lobe near the right kidney, and the left medial and left lateral lobes.



HORSES

Klebsiella spp. septicemia in horses associated with intravenous fluid administration

Murray Hazlett, Margaret Stalker, Tony van Dreumel, Bruce Duncan, Durda Slavic

Klebsiella oxytoca and *Klebsiella pneumoniae* are periodically isolated from cases of abortion, neonatal septicemia, and pneumonia. *K. oxytoca* septicemia has been reported as a cause of fatal septicemia in racehorses due to a unopened but contaminated commercial batch of electrolyte/amino-acid solution. Since 2009, we have had 4 horses from 3 premises submitted for postmortem examination that died of *Klebsiella* spp. septicemia, likely associated with improper preparation and storage of electrolyte/amino acid solutions.

In the first submission, 2 young Thoroughbreds were routinely jugged with a preparation of electrolyte solution, iron, copper, and multivitamins. One hour after administration to 1 horse, it was found with its head hanging and in distress; it developed seizures later that day and died. At postmortem, there were petechial hemorrhages on serosal surfaces of lungs, heart, and intestines. Microscopically, there were multifocal hemorrhages with widespread intravascular thrombosis in multiple organs. *K. pneumoniae* was isolated from the spleen and kidney of this horse, as well as from one of the jugs used in treating the horses. Large numbers of other bacteria, mostly coliforms, were also isolated from the residual fluid in the submitted jugs. A barn-mate had similar clinical signs, and also had lesions of bacterial septicemia at postmortem. It was severely autolyzed and, although coliform bacteria and *Streptococcus zooepidemicus* were isolated, *Klebsiella* spp. was not recovered.

The third horse was a young Standardbred that died within 24 hours of receiving lactated Ringer's solution with added vitamins, as well as a glyceryl guaiacolate jug. At postmortem, there was severe hemorrhage in fascia and muscles in the neck, lungs, heart and spleen (Figure 1). *Klebsiella oxytoca* was isolated from kidney, spleen and lung in large numbers, with lesser numbers of *Streptococcus zooepidemicus*. Mixed cultures of bacteria were recovered in the remnants of fluid in the treatment bottles, although

Klebsiella spp. was not specifically identified.

The fourth horse was a 5-year-old Standardbred that was administered a home-mixed vitamin jug in the morning and "appeared to react". The horse initially responded to epinephrine and steroids but then deteriorated and died that night. Lesions seen were similar to the previous horses. *Klebsiella oxytoca* was recovered in moderate numbers from lung, and large numbers from spleen. *E. coli* was also isolated from spleen.

In all 4 horses, lesions seen were typical of disseminated intravascular coagulation, compatible with *Klebsiella* spp. or other gram-negative septicemia.

Contamination of the administered solutions may be from contamination of any of the multi-dose vials used, improper handling, poor aseptic techniques when mixing the solutions, poor storage with inadequate refrigeration, or reuse of needles, intravenous sets, or jug bottles without proper sterilization between uses. Storage of the solutions after the initial contamination and without refrigeration would allow the introduced bacteria to proliferate to large numbers. AHL

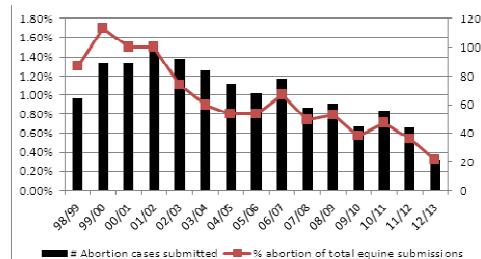


Figure 1. Severe acute subserosal splenic hemorrhage associated with *Klebsiella oxytoca* septicemia in a horse.

Equine abortion submissions are the lowest in 15 years

Beverly McEwen

Equine abortion submissions to the AHL are the lowest in 15 years, in absolute numbers and as a proportion of total equine cases received at the AHL. The reason for this dramatic decrease of submissions, which are less than half of the previous fiscal year, is not known as there are many variables including the economy that impact submissions to the diagnostic laboratory. Since 2006/2007, the proportion of non-infectious diagnoses of abortion, such as umbilical torsions and non-inflammatory placental lesions, has been greater than viral and bacterial diagnoses combined. Fiscal 2012/2013 is notable as the first year that there have been no cases of *Equid herpesvirus 1* (EHV-1) associated abortion identified since 1996/1997. This could be due to the absolute decrease in abortion submissions rather than a decrease in the prevalence of EHV-1 abortion. The 3:1 ratio of abortions submitted from Thoroughbred to Standardbred horses has not changed substantially over time. AHL



COMPANION ANIMALS

Necrotizing fasciitis due to *Streptococcus canis* infection in a dog

Andrew Brooks, John Prescott

A 3-year-old neutered male Golden Retriever that was hit by a car underwent orthopedic surgery to repair a fractured ilium. Shortly after the surgery, the dog was presented with fever, pitting edema around the surgical site, and a rapid onset of septic shock that resulted in death.

At postmortem examination, there was severe swelling of the left and right gluteal regions and diffuse swelling of the left hindlimb. Upon incising the skin of the left hindlimb, copious amounts of turbid red-brown exudate poured from the subcutaneous tissue and skeletal muscles (Figure 1). This exudate was present throughout the subcutaneous tissue and fascia of the left hindlimb and was accompanied by marked edema and hyperemia. Similar exudate surrounded the repaired ilium. The gross lesions indicated acute bacterial sepsis.

Histologically, the fascial connective tissues and skeletal muscles of the left hindlimb were marked by extensive necrosis, edema and suppurative inflammation. There was also myocarditis, meningitis, pulmonary alveolitis and microvascular thrombosis, which suggested that the dog had developed septicemia and a coagulopathy. Bacterial culture of the exudate from the left hindlimb isolated large numbers (4+) of *Streptococcus canis*. **The gross and histological lesions and the bacteriology results confirmed the diagnosis of necrotizing fasciitis due to *S. canis*.**

Necrotizing fasciitis (NF) and **streptococcal toxic shock syndrome (STSS)** are uncommon, severe, and potentially fatal conditions associated with invasive streptococcal infections. In dogs and cats, NF and STSS are usually due to infection with *S. canis*. Affected animals are often previously healthy but may have incurred an initiating injury, such as a bite wound or local infection. NF is characterized by necrosis and inflammation in connective tissue (fascia) with severe pain, fever and rapid clinical deterioration. STSS is characterized by hypotensive shock with multiple organ dysfunction and may overlap with NF. Bacterial superantigens, which bind to MHC class II molecules and T-cell receptors, are thought to be central to the pathogenesis by stimulating excessive release of pro-inflammatory cytokines. A novel bacterial virus-encoded mitogen resembling a superantigen has been identified in *S. canis* isolates; the lysogenic virus can be induced into the lytic state by treatment of the bacterium with fluoroquinolone antibiotics, and results in marked

expression of the mitogen. **It is important to note that treatment of dogs with fluoroquinolone antibiotics (FQ) early in the course of infection has been associated with the development of NF/STSS.** The dog in this case was treated with FQ, but the timing of therapy did not suggest a role in the pathogenesis. Veterinary staff should also be aware of the zoonotic risk posed by accidental cuts while debriding NF lesions. AHL



Figure 1. Severe inflammation and swelling of the fascial connective tissues of the hindlimb. Note the exudate pouring from the tissue.

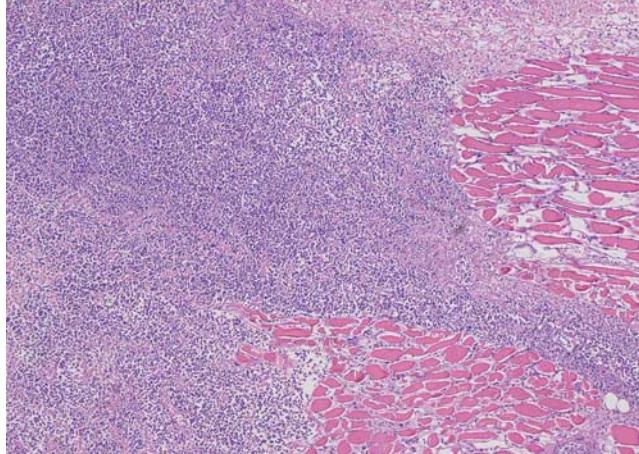


Figure 2. Histology of the left hindlimb. Severe necrosis and suppurative inflammation in the muscle and connective tissue (fasciitis).